

Claims

1. A method for inhibiting tumor cells, while reducing the risk of UV radiation exposure or vitamin D toxicity, said method comprising the step of administering to a patient a composition comprising an effective amount of a metabolic precursor of 1,25-dihydroxyvitamin D to increase levels of 1,25-dihydroxyvitamin D in said tumor cells in a target organ wherein the tumor cells have a hydroxylase enzyme for synthesizing 1,25-dihydroxyvitamin D from said metabolic precursor.
2. The method of claim 1 wherein said metabolic precursor is 25-hydroxyvitamin D.
3. The method of claim 1 wherein said hydroxylase enzyme is 25-hydroxyvitamin D-1 α -hydroxylase.
4. The method of claim 1 wherein said tumor cells are selected from the group consisting of prostate cancer cells, breast cancer cells, skin cancer cells, pancreatic cancer cells, colon cancer cells, lung cancer cells, leukemia cells, and lymphoma cells.
5. The method of claim 1 wherein said tumor cells are prostatic cancer cells.
6. The method of claim 1 wherein the effective amount of said metabolic precursor is an amount which results in intra-target organ cell levels of said metabolic precursor between about 25 and about 250 nmol/L.
7. The method of claim 1 wherein said metabolic precursor is administered as a composition comprising said metabolic precursor, or a salt, isomer, or derivative thereof, and a pharmaceutically acceptable carrier.
8. A method for inhibiting cancer cells, while reducing the risk of UV radiation exposure or vitamin D toxicity, said method comprising the step of administering to a patient a composition comprising an effective amount of a metabolic precursor of 1,25-dihydroxyvitamin D to increase levels of 1,25-dihydroxyvitamin D in cancer cells in a target organ wherein the target organ cancer cells have a hydroxylase enzyme for synthesizing 1,25-dihydroxyvitamin D from said metabolic precursor.

9. The method of claim 8 wherein said metabolic precursor is 25-hydroxyvitamin D.
10. The method of claim 8 wherein said hydroxylase enzyme is 25-hydroxyvitamin D-1 α -hydroxylase.
11. The method of claim 8 wherein said cancer cells are selected from the group consisting of prostate cancer cells, breast cancer cells, skin cancer cells, colon cancer cells, pancreatic cancer cells, lung cancer cells, leukemia cells, and lymphoma cells.
12. The method of claim 8 wherein said tumor cells are prostatic cancer cells.
13. The method of claim 8 wherein the effective amount of said metabolic precursor is an amount which results in intra-target organ cell levels of said metabolic precursor between about 25 and about 250 nmol/L.
14. The method of claim 8 wherein said metabolic precursor is administered as a composition comprising said precursor or a salt, isomer, or derivative thereof, and a pharmaceutically acceptable carrier.
15. A method for treating benign prostatic hyperplasia in an animal, while reducing the risk of UV radiation exposure or vitamin D toxicity, said method comprising the step of administering to the animal a composition comprising an effective amount of a metabolic precursor of 1,25-dihydroxyvitamin D to increase levels of 1,25-dihydroxyvitamin D in prostatic cells having a hydroxylase enzyme for synthesizing 1,25-dihydroxyvitamin D from said metabolic precursor.
16. The method of claim 15 wherein said metabolic precursor is 25-hydroxyvitamin D.
17. The method of claim 15 wherein said hydroxylase enzyme is 25-hydroxyvitamin D-1 α -hydroxylase.

18. The method of claim 15 wherein the effective amount of said metabolic precursor is an amount which results in levels of said metabolic precursor in prostate cells of between about 25 and about 250 nmol/L.

19. The method of claim 15 wherein said metabolic precursor is administered as a composition comprising said precursor or a salt, isomer, or derivative thereof and a pharmaceutically acceptable carrier.